The Law of the Heart.[1]

By Prof. E. H. Starling, C.M.G., F.R.S.

THE discovery by Harvey of the circulation of the blood, and of the part played by the heart in carrying on this circulation, is one of the few scientific discoveries which have become common knowledge. We have to think of the body as a collection of mechanisms or machines, each one of which is doing some form of work for one common end—*i.e.* the preservation of the body. For this work the oxidation of the food taken in at intervals during the day provides the energy; thus each part of the body must be supplied not only with food derived from the alimentary canal, but also with the oxygen taken in with the air we breathe into the lungs. Like any other machine, each body mechanism produces, as a result of this consumption of the food, waste gases and other waste products which have to be carried to the lungs or to the kidneys and there cleared out of the body. It is for this reason that the existence of the higher animal demands a common fluid, the blood, which can carry food, oxygen or carbonic acid, and is maintained in continual circulation between all the organs of the body, so that the alimentary canal, for instance, may serve for the maintenance of all parts, and the lungs can supply oxygen to these parts or excrete the carbonic acid which is produced as a result of their activity.

But a uniform mechanical circulation would be of little value to the body, since the activities of all its parts vary within wide limits. Thus, during muscular exercise the activity of the muscles may be increased tenfold or more, and this increase means a corresponding augmentation in their call for oxygen and in the quantity of waste products, especially carbonic acid, that they produce. Since the oxygen is carried by the blood, it follows that for the continued functioning of the muscles these must receive a blood supply which is ten times greater during activity than during rest if their activity is not to be brought to an end by a species of suffocation. Therefore, in any violent exercise involving the greater number of the muscles of the body, the circulation must be increased in force seven to ten times, and the heart, which is the pump maintaining the circulation, must under these conditions do from seven to ten times as much work as during rest.

The Mechanism of Adaptation.

What is the mechanism of these adaptations? How is it that the heart is able to carry on a circulation which may vary from a passage of 3 litres of blood per minute up to 30 litres of blood per minute (these figures representing the extreme limits between which the output of the heart-pump may vary according to the condition of the body)? It might be thought that we are dealing here simply with the influence of the central nervous system, which adapts the activity of the muscles of the body to the requirements of the environment, and that the heart being a muscle would be stimulated to contract more strongly at the same time as the nervous system calls into activity the voluntary muscles of the body. There is no doubt that the heart is under the control of the central nervous system, so that its action can be altered, increased, or diminished by the brain in accordance with the needs of the economy, but in the heart we find also a wonderful power of adaptation to the varying requirements of the organism which is quite independent of the central nervous system.

This can be shown quite easily either in the cold-blooded or warm-blooded animal. The heart of the frog and tortoise can be cut out and will continue beating for hours or even days. It has long been known that the heart of the mammal would beat for some minutes after being cut out of the body, but if we take pains to ensure that the muscles constituting the walls of the heart continue to receive their supply of oxygenated blood, the mammalian heart can be made to beat for eight to twelve hours after the death of the animal from which it is taken. In order to investigate this properly we want to make such a preparation that we can control at will all the conditions which may affect the action of the heart—viz. the amount of blood flowing into the heart from the big veins, the resistance which the heart has to overcome when it drives the blood out into the arteries, and the temperature at which the heart contracts. We must be able to measure at any time the output of the heart, the arterial pressure it maintains, its changes in volume during contraction, the pressure in all its cavities during contraction, the amount of blood flowing through the blood vessels of its walls, and its chemical exchanges, as measured by the amount of oxygen which it takes up and the amount of carbonic acid which it produces. It is these chemical changes which give the energy for the work of the heart.

The Heart-Lung Preparation.

All these procedures and controls can be carried out in the heart-lung preparation. In this preparation the pulmonary circulation from right ventricle to left auricle is left intact, and by means of artificial respiration the lungs are blown up rhythmically so that the blood in its course may take up oxygen and get rid of carbonic acid. The whole systemic circulation is replaced by rubber tubes. A glass tube is tied into the largest branch of the aorta, all the other branches being tied, so that the blood driven out by the left ventricle can escape only by the glass tube. From the glass tube a rubber tube passes to a thin rubber tube container within a wide glass tube. This thin rubber tube can be compressed to any desired extent by pumping air at a known pressure into the glass tube surrounding it. We can thus vary at pleasure the resistance which has to be overcome by the left ventricle, and, by maintaining a normal pressure in the beginning of the aorta, ensure a proper supply of oxygenated blood through the coronary arteries to the muscular tissue of the ventricles. It is this fact which makes it possible for the warm-blooded heart to continue to beat for eight to twelve hours after removal from the body. On the other side of the artificial resistance the blood is led through a spiral immersed in warm water to keep the blood at body temperature, and then passes into a reservoir from which a wide rubber tube leads to a glass tube placed in the big vein opening into the right auricle. By means of a screw clip on this tube the inflow of blood may be regulated to any desired extent, and can be kept constant while other conditions are varied. Thus in this preparation the three chief factors, temperature, the inflow of blood, and the resistance to the outflow of blood, can be varied separately and at the will of the operator. Any of the heart cavities or any part of the circuit can be connected to manometers so as to record the pressure of the fluid, and by means of a side tube placed just beyond the artificial resistance we can allow the blood to flow off into a graduated cylinder, and thus measure the time taken by the left ventricle to expel 50 or 100 c.c. of blood, thus measuring the average output of the organ.

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An Experiment Described.

A typical experiment may be divided into six stages. Records of one experiment show that in the first stage the heart was beating at a normal rate (72 per minute), the blood pressure varied from 100 mm. Hg, and the output of the heart was 240 c.c. of blood per minute. In the second stage the resistance to the flow of blood through the tubes was increased to such an extent that the pressure rose to 160–180 mm. Hg. The heart continued to beat, and for a time put out just as much blood as it did at the lower pressure. In the third stage the artificial resistance was suddenly reduced to zero, the arterial pressure fell to about 20 mm. Hg, but the heart beat regularly and the outflow of blood was unaltered because the inflow of blood had not been altered. In the fourth stage the inflow of blood was raised suddenly to 600 c.c. per minute. The heart became bigger, but the regularity of its contractions remained unaltered, and it drove forward all the blood that it received.

The same thing happened in the fifth stage, in which the artificial resistance was raised simultaneously with the venous inflow. The reason for these phenomena is that within certain limits the heart isolated from the body can respond to all the demands made upon it; it can overcome a higher resistance, and it can pump out more fluid. In the sixth stage the inflow of blood was further increased to 1200 c.c. per minute, and the artificial resistance was increased until the blood pressure rose to 200 mm. Hg. This was too much for the heart, which began to beat irregularly and dilate widely. It would have failed altogether if the pressure surrounding the thin rubber tube had not then been released to allow the artificial pressure to drop to a level at which the left ventricle could empty itself.

If during this experiment the amount of oxygen taken up by the blood had been measured, and also the amount of carbonic acid given off by this fluid in passing through the lungs, an increase in both these amounts would have been found during the stage at which greater demands were being made on the heart. That is to say, the greater the work done by the heart, the greater the chemical changes to supply energy. A motor-car may be running steadily with an even beat of its engines along a level road; when it comes to a hill it will slow up and finally stop unless the chauffeur increases the chemical changes and the energy of each explosion within the cylinder by opening the throttle and letting in more mixture of petrol and air. In the case of the heart there is no chauffeur, but there is some automatic regulation by which the heart increases its chemical changes, and therefore the energy of each beat, in exact proportion to the work which is demanded of it. It is the nature of this automatic regulation which concerns us now.

The Nature of the Automatic Regulation of the Heart.

By a careful observation of the changes in the heart in the experiment described above we may arrive at some clue to the nature of the pressure, but more accurate methods are necessary if we are to be certain of the correctness of our guess. We must, under these varying conditions, measure: (1) the pressure in the heart cavities produced at each contraction; (2) the volume of the heart cavities—*i.e.* the length of the muscle fibres of their walls. The first we measured in the experiment described by connecting the interior of each cavity in turn with a quickly acting manometer, the excursions of which are registered by an optical method so as to avoid the instrumental vibrations of a lever. The curve of pressure obtained under two conditions—*i.e.* low and high artificial resistance—could then be plotted. It must be remembered that the heart was sending on in each case all the blood that it received, though the work necessary under the high pressure was two or three times as great as that necessary to send on the blood at the low pressure. To measure the volume of the heart the ventricles are enclosed in an instrument known as a cardiometer. This communicates with a piston recorder so that the change of the volume of the ventricles at each beat can be registered on a moving surface.

The question we have to decide is: How does the heart know when it is relaxed that at the next contraction it will have to exert more force than it did previously, when the arterial resistance to be overcome was lower? If we measure the pressure in the ventricles in the manner just described we find that during the period of relaxation of the ventricles the pressure in its cavities is approximately zero, whether the artificial pressure which it has to overcome at its next beat is 50 or 150 mm. Hg. It is not, therefore, the tension on the walls of the heart which determines the strength of its contraction at its next beat. When, however, we come to measure the volume of the heart, we find that in the isolated heart this is directly proportioned to the work which the heart has to accomplish. Thus we find that the larger the heart—*i.e.* the more it is dilated during diastole—the greater is the pressure that it will get up at the succeeding contraction or systole.

We may put this in another form, as is shown by continuing our experiment over several hours, when we find that the worse the condition of the heart muscle, the more it must dilate in order to get up an adequate pressure. Other things remaining equal, we thus see that the volume of the heart during diastole is a measure of its physiological condition, and we are not surprised that a failing heart means a dilated heart. Of course there is a limit to this power of adaptation. As the heart dilates it is working at an ever-increasing mechanical disadvantage, and a point will finally arrive at which this disadvantage more than counterbalances the physiological effect of dilatation. The heart then dilates widely and fails to empty its contents. Dilatation of the heart means elongation of the muscular fibres composing its walls, so that we may put the law of the heart another way and say that the longer its muscle fibres the greater is the energy developed at each contraction. But in this form this wonderful power of adaptation possessed by the heart becomes part of the general properties of all muscular tissues, since the same rule applies to the fibres composing our voluntary muscles. Can we obtain any more precise and physiological conception of what is involved in this relationship between length of fibre and strength of contraction? Microscopic examination of the fibres, either of the heart or of voluntary muscle, shows that these are composed of innumerable fibrils, so that internally the muscle is made up of structures presenting an enormous extension of longitudinal surfaces. The more the muscle is stretched, the greater will be the extent of these surfaces. A large amount of evidence, based on the electrical and chemical changes occurring in muscle as a result of excitation, points to the contraction as being essentially a surface phenomenon—a molecular change over the whole of the longitudinal surface which may result in a polarisation or depolarisation of the surface and an increase of surface tension, so that the muscle is a surface tension machine in which there is on excitation a direct conversion of chemical into surface energy. The greater the surface the greater will be the number of molecules involved, so that increased length of muscle must increase at the same time the total chemical changes and the total tension produced by the summation of the surface tension of each fibril.

It is only by such a change of molecular dimensions that we can explain the rapidity of events in a muscle (the insect wing muscle can contract and relax 300 times per second), or the high efficiency of the machine, an efficiency which A. V. Hill has shown may amount to 100 per cent. for each isolated contraction, and over a length of time to 50 per cent. As directly measured in the heart-lung preparation, we find a mechanical efficiency of about 25–30 per cent.

Conclusion.

It is impossible here to enter into the applications of this law of the heart, but so far it has not failed in accounting for the behaviour of this organ under all manner of conditions, either in health or disease. It is important to remember, however, that we are dealing here with the isolated heart. In the natural body the mechanisms which we have studied are fenced round, protected and aided by the complex activity of the central nervous system, which is always acting on the heart, balancing its activity against that of the blood vessels, and co-ordinating it with the events which are occurring in every other part of the body. All these factors must be taken into account when we are endeavouring to form a conception of the total behaviour of this organ under the varying activities of the intact animal.

A Summer Visit to Jan Mayen Island.

By J. M. WORDIE.

JAN MAYEN ISLAND lies in 71° N. latitude, 8–9° W. longitude, and is approximately 300 miles north of Iceland, 200 east of Greenland, and 600 west and north-west respectively of Tromsö and Aalesund—the leading hunting ports in Norway. It was possibly discovered in 1607 by Henry Hudson and named "Hudson's Tutches"; the name, nevertheless, by which it is now known commemorates a Dutch seaman, Jan Jacobsz May, who visited the island in 1614. The evidence for the earlier visit by Hudson can scarcely be regarded as trustworthy. May's voyage, on the other hand, is well supported by documentary evidence. Immediately following its discovery, Jan Mayen became frequented almost every year by rival Dutch and British whalers. As a whaling and sealing centre, however, the island was markedly inferior to Spitsbergen. Its importance was, nevertheless, far from small, and the British Government is said to have made a grant of it to the Corporation of Hull in 1618. The number of whalers frequenting the island, however, dropped off very considerably about 1635, the immediate cause being probably a series of bad ice years.